cific position; for example, 2q11, 10q23 or 11q13. The frequency of autosomal fragile sites is about 0.2%. It has not been possible to link the fragile sites to any specific disease, except for fra(X)(q27) ("satellited X" or X-marker), which has proved to be associated with most cases of X-linked mental retardation.[1]

Fragile sites have some common features, some of them very different from any known chromosomal aberration. Their origin is unknown. We cannot exclude the possibility that fra(X)(q27) is not a cause but a symptom of a disease. Fra(X)(q27) may be caused by an error in folic acid metabolism. However, a similar specific chromosomal defect has been reported following a viral infection.[2]

Several diseases that were originally believed to be linked with a genetic factor — for example, kuru and Alzheimer's disease — are now thought to be caused by a transmissible agent.[3,4] Schizophrenia, another disease that affects the brain, is suspected of being of viral origin.[5] X-chromosome linkage is considered to exist in some forms of bipolar manic-depressive disease.[6] Therefore, we thought it possible that in patients with some of these diseases there would be fragile sites on an autosome or an X-chromosome.

To test the hypothesis that a presumed transmissible agent could damage a chromosome we performed the following preliminary experiments: The lymphocytes of a patient with fra(X)(q27)-positive mental retardation were cultured with cells from a patient who was negative for this marker. In another experiment the serum of a patient with this marker was added to the lymphocyte culture of a healthy man. In both experiments the marker was never induced in the fra(X)(q27)-negative cells. The lymphocytes of 4 patients with Alzheimer's disease, 11 with acute or chronic schizophrenia, 1 with unipolar psychosis and 1 with bipolar psychosis were cultivated in medium 199 (Gibco, Grand Island, New York) and checked for fragile sites. In no patients were fragile sites observed.

These negative results do not support the hypothesis that fragile sites originate from transmissible agents.

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Environmental lead and young children

To the editor: Dr. Fischel J. Coodin and his associates have added new elements to the discussion of environmental lead (*Can Med Assoc J* 123: 469, 1980). My editorial (122: 1347, 1980) was submitted in concern, not in complacency. Neither Schmitt and colleagues' paper (121: 1474, 1979) nor my editorial considered epidemic sources of lead.

Coodin and his associates referred to serious epidemic problems associated with lead. Specifically, they cited the dangers for young children of sniffing leaded gasoline. They also cited the excellent report by Settle and Patterson[1] that noted another way in which lead can be introduced into the population: consumption of tuna from cans sealed with lead. Coodin and his associates could have cited similar epidemics in history, including the celebrated 18th century Devonshire colic. Canada's record of preventing epidemics of lead poisoning has been good: it was quick to reduce the range of lead-based paints on the market; lead-sealed cans are very rare or nonexistent in Canada; and electric kettles constructed with lead solder have been withdrawn from the Canadian market.

As pointed out by Coodin and his associates, it is difficult to determine low concentrations of lead. Even more difficult is determining the effects of these low, and perhaps inaccurately measured, concentrations on the population, and particularly on young children.

The problem is twofold: how to accurately determine the situation in Canada regarding environmental lead, and how to remove the lead from the environment when necessary.

Coodin and his associates have added some valuable observations, and we are all indebted to Schmitt and colleagues for again bringing an important and continuing problem to the attention of the profession.

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Potassium iodide in the treatment of sarcoidosis

To the editor: Potassium iodide has been effectively used in the treatment of erythema nodosum.[1] Recently we successfully treated a case of sarcoidosis with this agent.

Case report

A 37-year-old man was referred to the National Defence Medical Centre in Ottawa for investigation of bilateral hilar adenopathy. He

was complaining of fatigue, general malaise, polyarthralgia and arthritis in both ankles of about 3 weeks' duration.

Physical examination revealed active arthritis in his left ankle and erythema nodosum over his right tibia. Laboratory values were within normal limits except for a slight increase in the serum levels of hepatocellular enzymes. A chest roentgenogram showed bilateral hilar adenopathy but no parenchymal disease. On the basis of the clinical presentation we felt confident in diagnosing sarcoidosis.[2]

The patient was initially treated with acetylsalicylic acid, 4800 mg/d, which resulted in some lessening of pain in the left ankle. This treatment was discontinued after 4 days and the patient began taking potassium iodide, 300 mg three times a day. Within 48 hours the pain, swelling and heat in the left ankle disappeared and there was accelerated regression of the erythema nodosum. The patient was discharged from hospital with no symptoms and was told to continue taking the potassium iodide for 6 weeks.

Discussion

Potassium iodide's immunologic effects are diverse,[3] but of particular note is its activity against granulomas.[2,3] Potassium iodide has been shown to decrease the size of non-infectious and infectious granulomas.[3] Osler and McCrae[4] in 1907 wrote that potassium iodide's "power to dissolve luetic tumors is the most dramatic thing in therapeutics". The pathogenesis of acute sarcoidosis is unknown, but granulomas are found in the synovium and overlying subcutaneous tissues of the affected joints. It is not unreasonable to suggest a causal role for granulomas in sarcoidosis.

The mode of action of potassium iodide against granulomas is unclear; however, one possibility is that this agent induces the release of heparin from sensitized mast cells.[1] Heparin and warfarin sodium appear to be able to suppress delayed hypersensitivity. Hedfors[5] reported a case in which pulmonary sarcoid regressed in a patient being treated for deep-vein thrombosis. Potassium iodide may be beneficial in patients with sarcoidosis or erythema nodosum or both.

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Pneumonia caused by *Neisseria meningitidis*, serogroup 135

To the editor: *Neisseria meningitidis* is an uncommon cause of pneumonia.[1] Patients with respiratory tract infection due to this organism resemble those with the more common meningococcal syndromes (i.e., meningitis and septicemia) in that most are young and otherwise healthy, and are often in military service.[2]

The following case is unusual on three counts: the patient was elderly and immunocompromised, and the organism was of an uncommon serogroup.

Case report

A 66-year-old woman was admitted to hospital with a pathologic fracture of the radius. She also had a 2-day history of cough with sputum production and chills. Rales and bronchial breathing were present bilaterally. A chest roentgenogram showed a right upper lobe infiltrate and right pleural effusion. Multiple myeloma was diagnosed on the basis of a bone marrow aspirate.

Gram-staining of the sputum revealed more than 25 pus cells and less than 10 epithelial cells per low power field and moderate numbers of gram-negative diplococci. *N. meningitidis* serogroup 135 was isolated from this specimen as well as from two blood cultures.

The pneumonia resolved completely with intravenous administration of penicillin G, 24 million units per day, for 2 weeks.

Discussion

Ten serogroups of *N. meningitidis* are now recognized: A, B, C, D, X, Y, Z, 135, Bo and 29E, although Bo appears to be identical to Y.[3] Group 135 was first identified in 1968,[3] but it has only recently been recognized as causing bacteremia,[4] septic arthritis,[4] pneumonia[4] and meningitis.[5,6] Pneumonia due to groups B, C, X or Z[2] and Y[2,7] has also been documented.

The only previous instance of pneumonia due to serogroup 135 was in a 4-month-old child.[4] I believe this is the first case reported in an adult.

I wish to thank the Public Health Laboratory, Toronto for confirmation and serogrouping of the *N. meningitidis* isolate.

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